

Bovine Dictyocaulosis: A Review

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Abstract: Dictyocaulus is a genus of very harmful parasitic roundworms that infects cattle, sheep, goats, horses and many other domestic and wild mammals. Husk, dictyocaulosis or parasitic bronchopneumonia is a serious parasitic disease in cattle caused by infection with the lungworm *Dictyocaulus viviparus* (*D.viviparus*). It is found worldwide, particularly in regions with temperate and cold climate. The epidemiology of lungworm disease is largely concerned with factors determining the number of infective larvae on the pasture and the rate at which they accumulate. Acute cases of the disease have coughing, respiratory distress, unthriftiness and rapid shallow abdominal breathing. Diagnosis can be done by taking history, clinical sign and faecal examination. Anthelmintics treatment with grazing management is the most to control dictyocaulosis.

Key words: *D. viviparus* • Dictyocaulosis • Larvae

INTRODUCTION

Production is a major agricultural activity with a huge economic impact in Ethiopia. An increase in large ruminant could contribute to the attainment of food self sufficiency in the country especially in requirement for the growing human population and to increase export earnings [1]. Infectious diseases of respiratory tract of farm animals are caused by combination of infectious agents and pre disposing factors such as inclement weather, stress of weaning, transportation and poorly ventilated housing, each of which can weaken defense mechanism of animals [2].

Dictyocaulus is a genus of very harmful parasitic roundworms that infects cattle, sheep, goats, horses and many other domestic and wild mammals. It is found worldwide, particularly in regions with temperate or cold climate. The most relevant species for livestock are: *Dictyocaulus filaria* infects mainly sheep and goats as well as camelids and other wild ruminants (Antelopes, deer, etc.). *D. viviparus* infects mainly cattle and other bovine, as well as camels, deer, elk, reindeer, etc. *Dictyocaulus arnfieldi* infects horses, donkeys, mules and other equine [3].

Bovine dictyocaulosis caused by the genus *D. viviparus* is the most common parasitic disease of cattle. Bovine parasitic bronchitis, or lungworm disease (“Hoose” or “husk”), is caused by the roundworm, *D.viviparus*. Infections with this parasite may occur in all ages of cattle, the disease is mainly seen in calves during their first season at grass. However, lungworm disease has recently emerged as a disease of second grazing season and older animals [4]. On most organic farms, a gradual infection occurs in young animals resulting in development of a natural immunity. However, on some farms this gradual infection does not take place and large numbers of infective larvae may build up on pasture. The challenge may be sufficient to cause clinical disease in cattle which have not developed adequate immunity [2].

The increase in bovine dictyocaulosis has been attributed to several factors, including climate change, a reduction in usage of the vaccine and/or common use of anthelmintic treatments to control lungworm and gastrointestinal parasites, which preclude adequate parasite antigen exposure, depriving the animal from subsequent immunological boosting. These challenges highlight the need for extensive research efforts to better understand the epidemiology and pathophysiology of

bovine dictyocaulosis, with the aim of developing improved therapeutic interventions [5]. Lungworm infection is one of the most important respiratory diseases of cattle, which is a roundworm (Nematode) parasite similar to gut worms. However, it completes its life cycle in the lungs rather than in the gastrointestinal tract [6].

Affected herds usually indicate high disease prevalence and mortality depending on the degree of pasture contamination [7]. Clinical signs in naturally affected animals are: loss of appetite, reduced growth, increase respiratory rate and coughing [8]. *D. viviparus* has been implicated as a parasite that causes high mortality in cattle [9]. Healthy animals get infection through intake of contaminated grass. Chronic inflammatory changes in infected lungs were in the form of ciliated epithelial cells loss, peribronchiolitis, eosinophilic bronchiolitis and atelectasis [10].

Dictyocaulus viviparus is one of the most common parasitic disease of cattle with a wide spread distribution in the temperate, subtropical and tropical climates. The lungworm (*D. viviparus*) is a relatively common parasite in tropical and subtropical area and causes heavy economic losses [11]. It is also found in the highlands of some African countries such as Congo, Kenya and Ethiopia [12].

Therefore, the objectives of this seminar paper are:

- To highlight the etiology, epidemiology, pathogenesis and clinical findings.
- To review possible treatment, control and prevention options.

Etiology: The nematode *D. viviparus* is the only lung worm of cattle. The disease it causes has many local names including: parasitic bronchitis, verminous pneumonia, verminous bronchitis and husk. Bovine lung worm has a very wide distribution through temperate and cold areas. The disease reaches its greatest importance in mild and damp regions of the British Isles and parts of Western Europe [4].

Morphology: Adult *Dictyocaulus* worms are slender, medium sized roundworms and up to 8 cm long. Females are about one third longer than males. They have a whitish to grayish color [13]. As in other roundworms, the body of these worms is covered with a cuticle, which is flexible but rather tough. The worms have a tubular

digestive system with two openings, the mouth and the anus [14]. They also have a nervous system but no excretory organs and no circulatory system, i.e. neither a heart nor blood vessels. The female ovaries are large and the uteri end in an opening called the vulva. Males have a copulatory bursa with two short and thick spicules for attaching to the female during copulation. The eggs of *D. viviparus* are ~35x85 micrometers, those of *Dictyocaulus filaria* and *Dictyocaulus arnfieldi* ~60x90 micrometers. They have ovoid shape and contain a fully developed L1 larva [3].

Epidemiology: Bovine parasitic bronchitis is a sporadic and largely unpredictable disease. This is because immunity develops more quickly than is the case with many other nematode infections, but nevertheless can remain incomplete for many weeks and may wane in the absence of re-infection. In most grazing seasons, immunity will develop fast enough to protect calves against the accumulating numbers of infective larvae on the grass. The farmer may not even realize that his land is contaminated. Clinical outbreaks occur when weather patterns, management or other factors result in sudden exposure to a pasture challenge sufficient to overwhelm any immunity that has already developed [4].

In comparison with the gastrointestinal nematodes of cattle, relatively few worms (i.e. a few hundred or thousand) are required to produce clinical signs. Thus, the disease is almost entirely confined to grazing cattle and occurs most frequently in young animals in their first year on grass, although outbreaks are becoming more common in adults. The epidemiology of lungworm disease is largely concerned with factors determining the number of infective larvae on the pasture and the rate at which they accumulate. Infective *D. viviparus* larvae are relatively inactive and are incapable of traveling more than 5 cm from the dung pat. Factors that disperse the larvae more widely over the pasture include mechanical spread by: Rain, Earthworms, Wheeled vehicles, Human and animal feet [4].

A fungus, *Pilobolus* plays a particularly important role in this process and can transfer larvae across field boundaries [13]. Fungal spores on grass pass through the grazing animal and germinate in the feces. *Dictyocaulus* larvae climb onto the sporangium (Fruiting body), which fills with water and bursts, propelling the fungal spore and the lungworm larvae for distances of up to 3 meter [12].

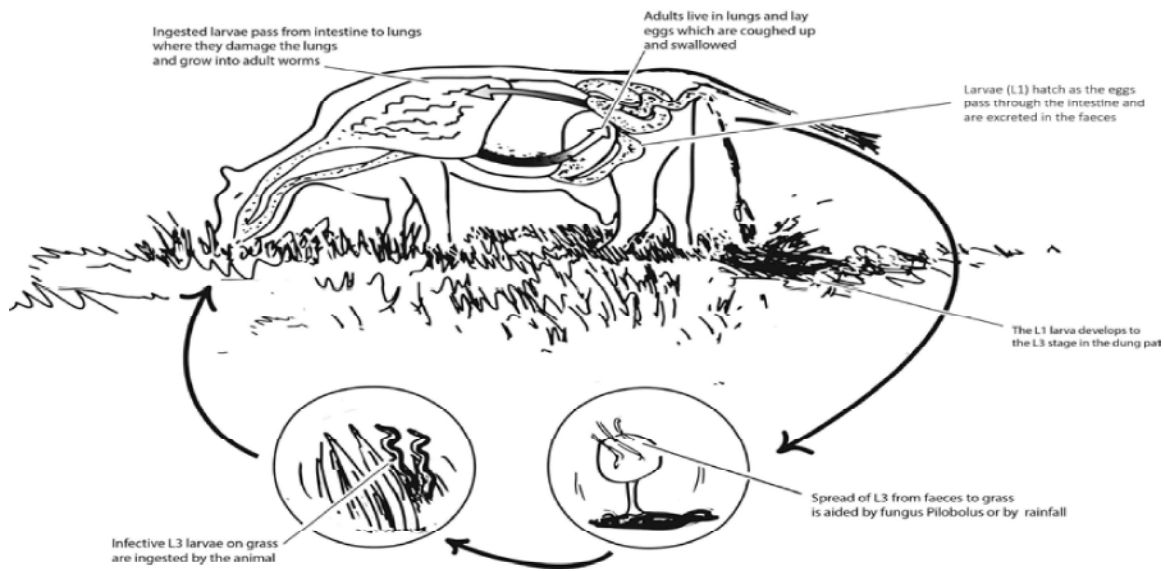


Fig. 1: *Dictyocaulus viviparous* life cycle [6].

Dairy calves are most vulnerable to lungworm disease as they are often reared indoors until 4-5 months of age and then placed on paddocks grazed each year by successive calf crops. If the paddocks are heavily contaminated, acute disease may occur in 1 week or so. However, only sufficient larvae survive the winter to induce low-grade asymptomatic infections in the susceptible calves, which then start to re-contaminate the pasture and recycle the infection. With the high stocking densities commonly used, pasture challenge can reach pathogenic levels within 2-4 months. This model does not satisfactorily explain all outbreaks and it has been suggested that larvae may be washed into the soil to emerge later [15].

In older animals larvae ingested in the autumn become hypobiotic and resume their development in the following spring. This event occasionally causes disease in housed cattle but such infections are usually asymptomatic and provide a source of pasture contamination when these carrier animals are put out to graze. This is thought to be the main source of infection in more severe climates where overwintering larvae may not survive on the pasture, but carrier animals have also been incriminated in disease outbreaks [4].

Life Cycle: The life cycle of *D. viviparous* is direct. Infection is by ingestion of third stage larvae (L3) from pasture (Fig.1). The L3 penetrate the intestinal wall and migrate via the lymphatic and vascular system, to reach the lungs approximately seven days after ingestion [16].

Here, the larvae migrate up the respiratory tree to the larger bronchi and trachea [17], where they mature to reproducing adult worms about 25 days after ingestion (This is the pre-patent period). Animals can harbor 100's-1000 of this white thread like worms and the adult females produce several thousand eggs that contain first stage larvae (L1). The eggs are coughed up and swallowed with mucus and the L1 hatch out during their passage through the gastrointestinal tract. The L1 are excreted in faeces where development to the infective L3 occurs, L3 subsequently leave the faecal pat via water or on the sporangia of the fungus *Pilobolus*. Infective L3 can develop within seven days of excretion of L1 in faeces, so that, under the appropriate environmental conditions, pathogenic levels of larval challenge can build up quickly [12].

Pathogenesis: Migrating *D. viviparous* larvae provoke little damage until they reach the lungs. Thereafter, passage of larvae up the bronchioles causes them to become blocked by mucus, eosinophils and other inflammatory cells, leading to collapse of the alveoli that they supply. Coughing and dyspnea occur if a sufficiently large volume of lung tissue is affected. This is accompanied by pulmonary edema and interstitial emphysema. As no structural damage has yet occurred, treatment at this stage in the disease produces an immediate clinical response. Later however when mature parasites are in the major bronchi, eggs and fragments of worms killed by immunity are aspirated and provoke foreign body pneumonia [4].

Secondary bacterial infections establish and cause bronchiectasis. Such lesions are slow to resolve and treated animals will require a long recovery period. Later still, once all or most of the worms have been expelled, the alveolar lining cells of some 25% of recovering animals become cuboidal and non-functional. The reason for this is unknown but may be a response to substances released by the dead worms. As this reaction is irreversible many animals affected in this way will die. The response of the lung varies widely depending on the number of larvae ingested, the nutritional status and age of the host and whether or not it is exposed to lungworm infection for the first time [6].

Vaccinated animals or those that have recovered from clinical or subclinical infection may cough and even become tachypneic if grazed on contaminated pasture. This is known as the re-infection syndrome and occurs as many larvae reach the lungs before succumbing to the immune response. Exposure of older previously infected animals to massive challenge may invoke a severe or fatal hypersensitivity reaction [4].

Clinical Findings: Outbreaks vary in severity from sporadic coughing with no apparent production loss to acute cases with a rapidly fatal outcome. Individuals within a group are usually affected to varying degrees. Poorly nourished animals appear less able to withstand lungworm infection. Nevertheless, it is not unusual for severe infestations to be fatal in well-fed calves. Acute cases have coughing, respiratory distress [18] and rapid shallow abdominal breathing of sudden onset that may reach a rate of 60-100 breaths/minute. There is a frequent "bronchial cough" a slight nasal discharge, a temperature of 40-41°C and a heart rate of 100-120 Beats per minute. The animal is bright and active and will attempt to eat, although respiratory distress often prevents this [4].

Progress of the disease is rapid and within 24 hour dyspnea may become very severe, accompanied by mouth breathing with the head and neck outstretched a violent respiratory heave and grunt, cyanosis and recumbence. On auscultation, lung consolidation is evidenced by loud breath sounds and crackles are heard over the bronchial tree. The crackling of interstitial emphysema commences over the dorsal two-thirds of the lung but is never as evident as in less acute cases. Fever persists until just before death, which usually occurs in 3-14 day and is greatly hastened by exercise or excitement [14].

Sub acute disease is more common in calves than the very acute form. The onset is usually sudden, the temperature is normal or slightly elevated and there is an

increase in the rate (60-70 breaths/minute) and depth of respiration. An expiratory grunt is heard in severe cases and expiration may be relatively prolonged. There are frequent paroxysms of coughing. The course of the disease is longer, 3-4 weeks and auscultation findings vary widely with the duration of the illness and the area of lung involved. In general, there is consolidation and bronchitis ventrally and marked emphysema dorsally. Affected animals lose weight very quickly and are very susceptible to secondary bacterial bronchopneumonia. The mortality rate is much less than in the acute form but many surviving calves have severely damaged lungs. Consequently, they may remain stunted for long periods and breathing may be labored for several weeks. Some surviving calves may show a sudden exacerbation of dyspnea around 7-8 weeks after the initial onset of disease. In these relapsed cases the prognosis is grave [4].

Adult dairy cattle are usually immune but sporadic outbreaks do occur due to waning immunity. Mortality is low but morbidity can be high with reduced milk yields causing significant economic loss. Coughing is a constant feature but other clinical signs are variable and may include dyspnea, nasal discharge and weight loss. Sudden exposure of immune adults to massive challenge can cause severe interstitial pneumonia [19].

Necropsy Findings: Adult *D. viviparous* is up to 8 cm long and easily seen when the trachea and bronchi are cut open. Worms may also be recovered by lung perfusion 12 up to several thousand may be present in severely affected animals. In pre-patent disease however careful microscopic examination of bronchial mucus is necessary to find larvae. Adult worms may be few or absent if the case is of sufficient duration for immune expulsion to have taken place [4].

In acute cases, morphological changes include: Enlargement of the lungs due to edema and emphysema [20], widespread areas of collapsed tissue of a dark pink color Hemorrhagic bronchitis with much fluid filling all the air passages Enlargement of the regional lymph nodes. Histologically, the characteristic signs are: Edema, Eosinophilic Infiltration, Dilatation of lymphatics and Filling of the alveoli and bronchi with inflammatory debris of Larvae in the bronchioles and alveoli [21].

In sub acute cases, interstitial emphysema is usually gross. Areas of dark pink consolidation are present in the diaphragmatic lobe and may also occur in other lobes. They can occupy two-thirds of the lung volume. There is froth in the bronchi and trachea. The regional lymph

nodes are enlarged. Histologically, eggs and larvae can be seen in the air passages, the bronchial epithelium is much thickened, the bronchioles are obstructed with exudates and the alveoli show epithelialization and foreign-body giant cell reaction. The re-infection syndrome is characterized by the presence of numerous 5 millimeter gray-green nodules formed by lymphoreticular cells clustering around dead larvae [22].

Diagnosis: Faecal samples can be submitted for analysis of L1 in Berman techniques [23]: a minimum of 15 gram faeces is required for this. However, it should be noted that false negative results can occur if the infection is still in the pre-patent period. Moreover, in adult cattle, which may have a degree of immunity, disease may occur in the absence of patent infection. For these reasons, laboratory confirmation of parasitic bronchitis by detection of L1 is only successful in a proportion of outbreaks [20]. An alternative to faecal analysis is to detect parasite-specific serum antibodies by enzyme linked immune sorbent assay (ELISA) [6]. In practical terms, when investigating an outbreak, it is advisable to analyze faecal and serum samples collected from a group of 6-10 animals that have been showing clinical signs of disease the longest. The ELISA is the preferred option when large numbers of samples are being tested [5].

Serological diagnosis has been evaluated in naturally and experimentally infected, as well as in vaccinated, animals. These studies have shown that positive ELISA titers are a satisfactory indicator of recent herd exposure, but they are not an accurate means of determining immune status of individual animals. The *D. viviparous* ELISA that is currently commercially available incorporates an antigen preparation that has been extracted from fifth larvae stage and adult worms [24]. In this test, early larval invasion following vaccination or during the pre-patent period is not detected and parasite specific serum antibody levels do not increase substantially until four to five weeks after initial challenge. Also, seroprevalence rates do not always accurately reflect the presence of clinical disease [25].

Many of the clinical signs of parasitic bronchitis are common to pneumonias of bacterial and viral origin. One feature which may be of value in differentiation is the relative softness and paroxysmal nature of the cough in parasitic infection. In adult cattle, the major problem in diagnosis is to differentiate the acute form of the disease from acute interstitial pneumonia due to other causes. Clinically, the diseases are indistinguishable and a history of movement onto a new pasture 1-2 weeks before the

onset of the disease may be common to both. It is necessary to demonstrate *D. viviparous* antibodies in blood, worms at necropsy and larvae in the herbage or in the feces of animals that previously grazed the pasture [4].

Treatment: The anthelmintics available for the treatment of bovine parasitic bronchitis are the modern benzimidazoles, levamisole and ivermectin. These drugs have been shown to be effective against all stages of lungworms with a consequent amelioration of clinical signs. In the past diethylcarbamazine was widely used, but it has been largely superseded by the drugs mentioned above [26]. For maximum efficiency all of these drugs should be used as early as possible in the treatment of the disease since clinical signs associated with pulmonary pathology are not rapidly resolved by mere removal of adult lungworms [19].

Where the disease is severe and well established in a number of calves, the stockowner should be warned that anthelmintic treatment, while being the only course available, may exacerbate the clinical signs in one or more animals with a possible fatal termination. The reasons underlying this are still under study, but are probably similar to those which produce post-patent parasitic bronchitis [13].

Whatever treatment is selected, it is advisable to divide affected calves into two groups as the prognosis will vary according to the severity of the disease. Those calves which are only coughing and/or tachypneic are usually in the pre-patent stage of the disease or have a small adult worm burden and treatment of these animals should result in rapid recovery. Calves in this category may not have developed a strong immunity and after treatment should not be returned to the field which was the source of infection; if this is impossible, parenteral ivermectin may be used since its residual effect prevents re-infection for a further three weeks. Any calves which are dyspnoeic, anorexic and possibly pyrexia should be kept indoors for treatment and further observation [13].

Control and Prevention: The best method of preventing parasitic bronchitis is to immunize all young calves with lungworm vaccine. This live vaccine, consisting of larvae attenuated by irradiation, is currently only available in Europe and is given orally to calves aged eight weeks or more [13]. Two doses of vaccine are given at an interval of four weeks and in order to develop a high level of immunity, vaccinated calves should be protected from challenges until two weeks after their second dose. Although vaccination is effective in preventing clinical

disease, it does not completely prevent the establishment of small numbers of lungworms [14]. Consequently, pastures may remain contaminated at a very low level. For this reason it is important that all of the calves on any farm should be vaccinated whether they go to pasture in the spring or later in the year [11]. Although, once a vaccination program has been undertaken it must be continuous for each calf crop. Although the limited pasture larval contamination will serve to boost the immunity of vaccinated calves it can lead to clinical disease in susceptible animals [13].

Control of parasitic bronchitis in first year, grazing calves has been achieved by the use of prophylactic anthelmintic regimens either by strategic early season treatments or by the administration of rumen boluses, as recommended in the control of bovine ostertagiosis. The danger of these measures however is that through rigorous control in the first grazing season, exposure to lungworm larvae is so curtailed that cattle remain susceptible to husk during their second season; in such situations it may be advisable to consider vaccination prior to their second year at grass [19].

Economic Importance of Bovine Dictyocaulosis: Severe *D. viviparus* infections can lead to complications that can cause a mortality rate of 20% or more among affected animals. Larval lungworms irritate the bronchioles before eggs can be seen in nasal secretions or larvae appear in feces. Later, the adult worms irritate the trachea and bronchi. In both stages, increased respiratory secretion causes lung congestion. The disease caused by lungworm is parasitic bronchitis, also called husk, which is characterized by rapid shallow breathing and coughing. Severe cases lead to emphysema and pneumonia – heavy infections can cause death [19].

This disease is recognized as a leading cause of economic losses in cattle farming – mainly in the temperate zones. Losses, due to bovine dictyocaulosis, can be direct (Death of calves, mainly during the first year of pasture) or indirect (Reduced performance, growth delay and treatment costs). Although much has been accomplished relating to bovine lungworm infection and despite the availability of effective anthelmintics and a vaccine, this disease continues to plague the cattle population as a burgeoning problem without any signs of abatement. There has been an increase in the incidence of husk in recent years; first season calves are particularly affected, although yearling and adult cattle may also succumb to the disease. Lungworm is responsible for reduced weight-gain and deaths in calves and yearlings and lowered milk-yield in dairy cows [3].

CONCLUSIONS AND RECOMMENDATIONS

Bovine dictyocaulosis caused by the species *D. viviparus* is the most common parasitic disease of cattle with a worldwide distribution. The epidemiology of lungworm disease is largely concerned with factors determining the number of infective larvae on the pasture and the rate at which they accumulate. Parasitic diseases have a great impact on large ruminant production. Bovine lungworm is recognized as a leading cause of economic losses in cattle farming mainly in the tropical and sub-tropical zones. In Ethiopia due to lack of enough control and prevention strategies the disease remain an endemic in most parts of the country. Losses, due to bovine dictyocaulosis, can be direct (Death of calves, mainly during the first year of pasture) or indirect (Reduced performance, growth delay and treatment costs). Therefore, based on the above conclusions the following recommendations are forwarded.

- ▶ Calves should be kept housed until they can be turned out onto pasture as one group.
- ▶ All ages of cattle are monitored over the grazing season in the early housing period.
- ▶ Anthelmintics treatment should always be combined with pasture management.
- ▶ Treatment of the whole group should be undertaken as soon as possible after the appearance of clinical signs in order to limit the impact of the infection.
- ▶ Applying rotational grazing system for different seasons of the year would apparently reduce pasture contamination. Therefore farmers should be informed to use this technique at least after harvesting their crop.

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